We claim:

1. An isolated nucleic acid molecule which encodes an ALK-1 protein, the complementary sequence of which hybridizes, under stringent conditions to the nucleotide sequence set forth in SEQ ID NO: 1.

2. The isolated nucleic acid molecule of claim 1, wherein said isolated nucleic acid molecule is cDNA.

3. The isolated nucleic acid molecule of claim 1, wherein said isolated nucleic acid molecule is genomic DNA.

4. The isolated nucleic acid molecule of claim 1, which encodes a protein whose amino acid sequence is the amino acid sequence encoded by SEQ ID NO: 1.

5. The isolated nucleic acid molecule of claim 1, consisting of SEQ ID NO: 1.

6. The isolated nucleic acid molecule of claim 1, comprising nucleotides 283 to 1791 of SEQ ID NO: 1.

7. Expression vector comprising the isolated nucleic acid molecule of claim 1, operably linked to a promoter.

8. Recombinant cell comprising the isolated nucleic acid molecule of claim 1.

9. Recombinant cell comprising the expression vector of claim 7.

10. Isolated protein encoded by the isolated nucleic acid molecule of claim 1.

11. The isolated protein of claim 10, comprising the amino acid sequence of the protein encoded by SEQ ID NO: 1.

12. Antibody which binds to the isolated protein of claim 10.

13. The antibody of claim 12, wherein said antibody binds to an extracellular domain of said protein.

14. A method for inhibiting expression of a gene, expression of which is activated by phosphorylated Smad1 or phosphorylated Smad-5, comprising contacting a cell which expresses said gene and which presents ALK-1 on its surfaces with an inhibitor which interferes with phosphorylation of Smad1 or Smad-5.

15. The method of claim 14, wherein said inhibitor inhibits binding of TGF-ß to ALK-1.

16. The method of claim 14, wherein said inhibitor is an antibody which binds to TGF-ß.

17. The method of claim 14, wherein said inhibitor is an antibody which binds to an extracellular domain of said protein.

18. The method of claim 14, wherein said inhibitor inhibits binding of said Smad1 or Smad-5 to ALK-1.

19. The method of claim 18, wherein said inhibitor is Smad6 or Smad7.

20. The method of claim 14, wherein said inhibitor inhibits interaction of said Smad1 or Smad-5 with a type II, TGF-ß receptor.

21. A method for enhancing expression of a gene, expression of which is activated by phosphorylated Smad1 or Smad-5, comprising contacting a cell which is capable of expressing said gene with a molecule which activates phosphorylation of Smad1 or Smad-5.

22. The method of claim 21, wherein said molecule binds to the extracellular domain of ALK-1.

23. The method of claim 21, wherein said molecule is TGF-ß.

24. The method of claim 21, wherein said molecule is a portion of TGF-ß sufficient to bind to ALK-1.

25. The method of claim 21, wherein said molecule phosphorylates Smad1 or Smad-5 without interaction with ALK-1.

26. The method of claim 21, wherein said molecule facilitates interaction of ALK-1 and a TGF-ß type II receptors.

27. A method for determining if a substance effects phosphorylation of Smad1 or Smad-5, comprising contacting a cell which expresses both Smad1 and ALK-1, or both Smad-5 and ALK-1 with a substance to be tested and determining phosphorylation of Smad1 or Smad-5, or lack thereof.

28. A method for identifying a gene whose activation is effected by phosphorylated Smad1 or phosphorylated Smad-5, comprising contacting a first sample of cells which express and phosphorylate Smad1 or Smad-5 with an agent which inhibits or activates phorphorylation of Smad1 or Smad-5, removing transcripts of said cell sample, and comparing said transcripts from transcripts of a second sample not treated with said agent, wherein any differences therebetween are transcripts of genes whose activation is effected by phorphorylation of Smad1 or Smad-5.